treatment, and 88 mm Hg after six months' treatment ($p>0·5$, paired t test; 95% confidence interval for the difference between means −3 to + 4 mm Hg). Similar stability was seen in the placebo group, in whom the corresponding diastolic pressures were 93, 92, and 92 mm Hg ($p>0·30$, paired t test; 95% confidence interval −5 to + 2 mm Hg). After six months' treatment the change in blood pressure from the pretreatment value was not significantly different between the two groups (diastolic: $p=0·4$ (unpaired t test), 95% confidence interval for the difference between means −3 to + 7 mm Hg; systolic: $p=0·9$ (unpaired t test), 95% confidence interval −9 to + 8 mm Hg).

There was no significant difference in serum magnesium, potassium, sodium, or creatinine concentrations between the two groups after six months' treatment ($p>0·05$, unpaired t test).

Comment

In spite of the fact that our trial was designed to detect a minimal relevant difference of 7 mm Hg in diastolic pressure at the 5% significance level with a power of 85% and an estimated standard deviation of 7 mm Hg[2] we were unable to confirm Dyckner and Wester's finding of an apparent hypotensive effect of magnesium supplementation. Our trial indicates that magnesium supplementation does not exert a clinically important effect on blood pressure when given to hypertensive patients receiving long term diuretic treatment.

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1 Dyckner T, Wester PO. Effect of magnesium on blood pressure. *Br Med J* 1983;**286**:1847-9.
2 Schødt T. How many patients are needed for a clinical trial? *Ugeskr Laeger* 1985;**147**:3232-3.

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A simple grading system to guide the prognosis after hip fracture in the elderly

Kellogg Speed described fracture of the neck of femur as "the unsolved fracture," and much work has been carried out to find the surgical solution to this fracture. The problem is not simply a surgical one, however, and the social implications should not be underestimated. Miller suggested that fracture of the hip may be viewed as a disease with a predictable rate and pattern of mortality.[3] The importance of social circumstances and state of general health at the time of fracture in influencing the rehabilitation of elderly patients with hip fractures has been shown.[1-3] Predictive studies of rehabilitation after hip fracture are scarce, and those which have been published fail to offer an uncomplicated bedside grading system suitable for routine use at the time of admission.[3-5] In a prospective study of 322 patients we have tested a simple bedside formula for use as a guide to prognosis after fracture of the femoral neck.

Patients, methods, and results

A prospective study of 322 patients over the age of 65 with fractures of the femoral neck (intracapsular and extracapsular) was carried out. Patients were assessed by one of the authors before surgery and allocated points according to social circumstances and medical state. For social circumstances those who were totally independent were given one point, those living alone with help two points, and those living in an institution three points. For medical state those in good general health were given one point, those with satisfactory general health but a history of previous serious illness two points, and those with poor health three points. The scores from both categories were added, giving each patient a possible combined score of two to six. Six months after surgery the patients' circumstances were reassessed by one of the authors. They fell into one of three unambiguous categories: satisfactory (back to original circumstances within six weeks of fracture), poor (still in hospital six weeks after fracture with no immediate prospect of return to previous circumstances), and dead.

Six months after fracture 48 (15%) of the patients had died. This compares favourably with other studies.[1] The table compares the numbers of patients with satisfactory results, patients with poor results, and those who died according to total score.

Outcome of treatment according to points scored at initial assessment

	Score at initial assessment				
	2 (n=109)	3 (n=81)	4 (n=88)	5 (n=29)	6 (n=15)
Outcome of treatment (% of patients):					
Satisfactory	93	79	20	0	0
Poor	4	11	69	59	13
Died	3	10	11	41	87

Comment

The most important finding of this study was that no patient with a score of 5 or 6 had a satisfactory result. Those with scores of 3 or less can be expected to make satisfactory progress, while those with scores of 5 or 6 are unlikely to return to their circumstances before the fracture. Thus an elderly person living either on her own or in an institution who is in poor health is unlikely to return to her original circumstances. With vigorous rehabilitation the outcome for those with a score of 4 may be improved. At a time of limited resources the question arises as to whether those with scores of 5 or 6 should be placed in long stay care immediately after surgery, thus freeing the rehabilitation team to concentrate on those with a prospect of satisfactory recovery. This simple scoring system is easy to remember and can give an instant bedside guide to prognosis after hip fracture.

1 Miller CW. Survival and ambulation following hip fracture. *J Bone Joint Surg (Am)* 1978;**60A**:730-934.
2 Ceder L, Throngren K-G, Wallden B. Prognostic indicators and early home rehabilitation in elderly patients with hip fractures. *Clin Orthop* 1980;**152**:173-84.
3 Carrol D. Prediction of performance of patients with hip fracture. *Md State Med J* 1969;**18**:69-74.
4 Ceder L. Statistical prediction of rehabilitation in elderly patients with hip fractures. *Clin Orthop* 1980;**152**:185-90.
5 Cobey JC, Cobey JH, Conant L. Indicators of recovery from fractures of the hip. *Clin Orthop* 1976;**117**:258-62.

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Severe cutaneous reactions to alternative remedies

"Alternative" or "complementary" treatments are widely employed by patients, often without medical advice. Three cases are described of severe cutaneous reactions to alternative remedies.

Case reports

Case 1—A 46 year old man presented with erosion and blistering of the mouth, lips, and penis and soreness and redness of the eyes. The appearance was that of the Stevens-Johnson syndrome and he was admitted to hospital, where he required systemic steroid treatment. He gave no history of preceding illness or infection and was taking no medications. He did, however, admit that one week before the onset of the eruption he had visited a health food shop looking for a general tonic; he had purchased two bottles of tablets, one unlabelled, subsequent analysis of which yielded only lactose, and the other called "Golden Health Blood Purifying Tablets": these contained extracts of red clover, burdock, queen's delight, poke root, prickly ash, sassafras bark, and *Passiflora*. He admitted that some months previously he had had a similar, but rather less severe reaction a few days after taking these tablets. The patient's condition resolved, but 10 months later he returned with a clinically identical eruption, again after the use of Golden Health Blood Purifying Tablets.

Case 2—A 56 year old man presented with an acute generalised pusular psoriasis. He gave a 25 year history of chronic plaque psoriasis, which had been

well controlled with topical agents until four days earlier, when he had purchased a 1 kg packet of Dead Sea Mineral Salts, which he added to his bathwater. Within two days he had become generally unwell and his psoriasis erythrodermic. He required admission to hospital and treatment with methotrexate and oral retinoids. His psoriasis remained well controlled until two years later, when it was noted to be once again inflamed and unstable; on direct questioning he admitted that he had recently again tried the Dead Sea Mineral Salts, though on this occasion he had used only about 250 g per bath. The stated ingredients of the preparation used (Dead Sea Health Products, Kibbutz Ein Gedi MP, Dead Sea, Israel) included potassium chloride 22-28%. magnesium chloride 30-34%, bromides 0·2-0·4%, and sulphates 0·1-0·2%.

Case 3—A 22 year old woman with lifelong mild topic eczema visited a naturopath, not medically qualified, who recommended an injection of a homoeopathic remedy, Nat Mur 200. Within 12 hours she was in an acute erythrodermic state, requiring intensive medical treatment. The mechanism of this reaction was obscure, since homoeopathic remedies are not normally administered by injection and are usually in such a highly diluted form as to contain no measurable quantity of the original ingredients.

Comment

Alternative or complementary medicine is widely used for a range of disorders. Many patients find benefit, and though few of these therapies have been subjected to the same critical evaluation of safety and efficacy as conventional medicines, even the most sceptical of doctors generally regard them as harmless; indeed, it may well be the apparent safety of these forms of treatment which contributes to their popular appeal.

These three cases illustrate severe cutaneous reactions to different sorts of alternative medicines. The prevalence of such adverse reactions is impossible to assess, since there is no central monitoring authority equivalent to the Committee on the Safety of Medicines; furthermore, patients may be reluctant to admit to their doctor that they have employed a non-medical remedy. Alternative medicines are also not subject to the same requirements of standardisation as pharmaceuticals.

Many forms of complementary medicine are of potential value. Nevertheless, it is also important to be alert to their potential hazards. A valuable first step would be a central monitoring body.

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Effect of milk on patients with duodenal ulcers

A diet with a high milk content is often advised for patients with a duodenal ulcer, perhaps because milk is effective at neutralising acid.[1] Moreover, epidemiological data suggest that the incidence of peptic ulcers is inversely related to milk consumption.[2] In contrast, a diet with a high milk content stimulates significantly greater acid production than a normal diet[3]; its value to patients with duodenal ulcers has therefore been questioned.[4] The effect of milk on the healing of duodenal ulcers has not, however, been clearly established, so we performed a controlled therapeutic trial on patients admitted to hospital with ulcers.

Patients, methods, and results

Sixty five consecutive patients with duodenal ulcers confirmed by endoscopy were included in the study. On admission to this hospital they were randomly allocated to one of two groups, group I consumed a normal hospital diet, and group II received a diet consisting exclusively of milk (500 ml at breakfast, 750 ml at lunch, and 750 ml at dinner). They were allowed to add sugar according to their taste, and both groups were allowed to consume seasonal fruits. The total daily intakes of calories (1800-2000) and protein (60-70 g) in the two groups were very similar. Both groups received cimetidine 200 mg three times daily and 400 mg at bedtime, making a total of 1 g a day. Treatment continued for four weeks, after which patients underwent endoscopy again. The ulcers were then categorised as either healed, with or without residual duodenitis, or not healed (irrespective of size).

Three of 33 patients (9%) in group II developed milk intolerance in the form of diarrhoea and colicky abdominal pain and were excluded from the study. Weekly comparison of symptomatic relief showed no appreciable difference between the two groups throughout the study (table). Endoscopic assessment after four weeks of treatment showed that the proportion of healed ulcers was significantly higher in patients receiving a normal diet (78%) compared with those who consumed only milk (53%) ($p<0·05$). Serum calcium and creatinine concentrations were within the normal range in both groups before and after treatment.

Age of and clinical information on patients with ulcers participating in study

	Group I (normal diet)	Group II (milk diet)
Patients:		
Total	32	33
Dropped out	0	3
Completed treatment	32	30
Age (years):		
Mean (SD)	37·4 (9·3)	39·6 (9·9)
Range	22-60	24-70
Median	35	40
Ratio M:F	32:0	30:0
Duration of symptoms (years):		
Mean (SD)	5·2 (3·0)	6·1 (3·2)
Range	1-12	1-14
Median	5	6
No (%) of smokers	27 (84)	23 (77)
No (%) of patients whose symptoms were relieved:		
Week 1	11 (35)	11 (37)
Week 2	21 (66)	19 (63)
Week 3	24 (75)	21 (70)
Week 4	28 (88)	25 (83)
No (%) of patients whose ulcers healed as shown by endoscopy	25 (78)	16 (53)*

*$\chi^2=4·24$; $p<0·05$. There was no significant difference in the other parameters between the two groups.

Comment

In a previous study Doll *et al* found that a diet with a high milk content and even an intragastric milk drip did not influence the healing of peptic ulcers.[1] Their study, however, was based on a barium meal examination, which is not as accurate as endoscopy in assessing ulcer healing, especially in the presence of a deformed duodenal bulb. In our study the proportion of healed ulcers in patients on a normal diet was 78%, which is the usual response obtained with cimetidine. In contrast, there was a significant reduction in the proportion of healed ulcers in patients who received a diet with a high milk content. Pain relief in the two groups was very similar, and this is perhaps why milk has been found to be useful in treating duodenal ulcers.

The precise mechanism by which milk delays ulcer healing is conjectural. There is no evidence to suggest an interaction between milk and cimetidine. One possibility is that the high content of calcium in milk stimulates excess acid production. Extracellular calcium plays a part in gastrin mediated acid production, and patients with duodenal ulcers are more sensitive to this effect than normal subjects.[5] Calcium may not be the only factor, however, as even milk that is low in calcium produces a significantly greater increase in acid secretion in patients with duodenal ulcer than in normal subjects.[4]

We suggest that a diet with a high milk content has an adverse effect on the healing rate of duodenal ulcers and should not be recommended for these patients.

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1 Doll R, Friendlander P, Pygott F. Dietetic treatment of peptic ulcer. *Lancet* 1956;i:5-8.
2 Paffenbarger RS Jr, Wing AL, Hyde RT. Chronic disease in former college students. XIII. Early precursors of peptic ulcer. *Am J Epidemiol* 1974;**100**:307-15.
3 Bingle JP, Lennard-Jones JE. Some factors in the assessment of gastric antisecretory drugs by a sampling technique. *Gut* 1960;**1**:337-44.
4 Ippolite AF, Maxwell V, Isenberg JI. The effect of various forms of milk on gastric acid secretion. Studies in patients with duodenal ulcer and normal subjects. *Ann Intern Med* 1976;**84**:286-9.
5 Christiansen J, Kirkegaard P, Olsen PS, Peterson B. Interaction of calcium and gastrin on gastric acid secretion in duodenal ulcer patients. *Gut* 1984;**25**:174-7.

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